



3

[PubMed](#) [Nucleotide](#) [Protein](#) [Genome](#) [Structure](#) [PMC](#) [Taxonomy](#) [OMIM](#) [Bc](#)
Search
[Limits](#)[Preview/Index](#)[History](#)[Clipboard](#)[Details](#)[About Entrez](#)

[Text Version](#)[Entrez PubMed](#)[Overview](#)[Help | FAQ](#)[Tutorial](#)[New/Noteworthy](#)[E-Utilities](#)[PubMed Services](#)[Journals Database](#)[MeSH Database](#)[Single Citation Matcher](#)[Batch Citation Matcher](#)[Clinical Queries](#)[LinkOut](#)[Cubby](#)[Related Resources](#)[Order Documents](#)[NLM Gateway](#)[TOXNET](#)[Consumer Health](#)[Clinical Alerts](#)[ClinicalTrials.gov](#)[PubMed Central](#)[Privacy Policy](#)

1: *Acta Pharmacol Toxicol (Copenh)*. 1981 Nov;49(5):334-53.

[Related Articles](#)[Links](#)

Inhibition of adrenergic neuroeffector transmission in rabbit pulmonary artery and aorta by adenosine and adenine nucleotides.

Husted S, Nedergaard OA.

The effect of adenosine and adenine nucleotides on sympathetic neuroeffector transmission in the rabbit isolated pulmonary artery and aorta was studied. Adenosine ($10(-5)$ - $3 \times 10(-4)$ M) decreased the contractile response of pulmonary artery and aorta evoked by electrical-field stimulation. The decrease was reversible. No tachyphylaxis developed. Inhibition of either adenosine deaminase by deoxycoformycin ($3.6 \times 10(-6)$ M) or of adenosine transport by dilazep ($3 \times 10(-6)$ M) did not alter the inhibitory effect of adenosine on the neurogenic contractions in the pulmonary artery. However, deoxycoformycin plus dilazep markedly enhanced the inhibitory effect of adenosine. The calcium antagonists nifedipine ($1.5 \times 10(-8)$ M) and nimodipine ($1.3 \times 10(-8)$ M) had no effect on the adenosine-induced inhibition. This was also the case with theophylline ($5 \times 10(-5)$ M), atropine ($10(-7)$ M), and the prostaglandin synthetase inhibitors indomethacin ($5 \times 10(-5)$ M) and suprofen ($3 \times 10(-5)$ M). The contractile response of the pulmonary artery elicited by exogenous (-)-noradrenaline (NA; $10(-9)$ - $3 \times 10(-4)$ M) was essentially not altered by adenosine ($10(-5)$ - $3 \times 10(-4)$ M). Adenosine ($10(-4)$ M) did not alter the spontaneous 3 H-outflow from rabbit aorta preloaded with 3 H-(-)-noradrenaline (3 H-NA). Adenosine ($10(-5)$ - $3 \times 10(-4)$ M), ADP ($10(-4)$ M), ATP ($10(-5)$ M), and inosine ($10(-4)$ M) diminished the overflow of tritium from pulmonary artery and aorta preloaded with 3 H-NA. The spontaneous outflow of tritium from aorta preloaded with 3 H-NA consisted of 3 H-NA (17%), 3 H-dihydroxyphenylglycol (3 H-DOPEG; 30%), 3 H-dihydroxymandelic acid (3 H-DOMA, 4%), 3 H-O-methylated and deaminated metabolites (3 H-OMDA; 42%), and 3 H-normethanephrine (3 H-NMN; 2%). Adenosine ($10(-5)$ and $10(-4)$ M) enhanced 3 H-DOPEG and 2 H-NMN, decreased 3 H-NA, and did not alter 3 H-DOMA and 3 H-OMDA. The stimulation-evoked overflow of tritium for aorta preloaded with 3 H-NA consisted of 3 H-NA (31%), 3 H-DOPEG (18%), 3 H-DOMA (2%), 3 H-OMDA (46%), and 3 H-NMN (3%). Adenosine ($10(-5)$ and $10(-4)$ M) enhanced 3 H-NA and 3 H-DOPEG, decreased 3 H-OMDA and did not alter

3H-DOMA and 3H-NMN. Adeosine (10(-6)-10(-4)M) did not alter the accumulation of 3H-NA (10(-8)M) by aorta. It is concluded that adenosine inhibits vascular sympathetic neuroeffector transmission by diminishing the release of transmitter from the nerve terminals.

PMID: 6285668 [PubMed - indexed for MEDLINE]

[Display](#) [Abstract](#)  Show: [20](#)  [Sort](#)  [Send to](#) [Text](#) 

[Write to the Help Desk](#)

[NCBI](#) | [NLM](#) | [NIH](#)

[Department of Health & Human Services](#)

[Freedom of Information Act](#) | [Disclaimer](#)

Jun 5 2003 10:08:34